TRAUMATISM AND HÆMATOMYELIA AS CAUSES OF SYRINGOMYELIA.[1]

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THE following case of fracture of the fourth cervical vertebra occurred in the service of Dr. Willard at the Presbyterian Hospital. We desire to thank him for permission to use it in this paper. The article will appear more in detail in the *International Medical Magazine* for April, but the present paper has been rewritten and much changed with the exception of the short histological report.

When examined microscopically the fifth cervical segment showed considerable alteration of tissue, above the lower part of the fourth and below the upper part of the sixth segment very little that was pathological could be noted. In the fifth cervical segment the fibres of the median portion of each lateral column were pressed from a longitudinal into a horizontal course; in the upper part of the segment each anterior horn was encroached upon at its posterior border by these fibres; slightly lower the interior of the right anterior horn was filled by a mass of fibres from the antero-lateral column, and was surrounded at all points by a hemorrhagic ring of gray matter; still lower in the segment the anterior border of each anterior horn was broken through by penetrating fibres. The force seemed to have been exerted at the centre of the cord and from the front, probably from pressure of the fifth cervical vertebra, and it appeared as though a wedge of fibres had been driven into each anterior horn from the white matter. The hemorrhage in the white matter was not great at any point; throughout the destroyed area there was extensive hemorrhage in both anterior and posterior horns, leaving the commissures free, and this was greatest at the upper part of the sixth segment, where the fibres of the white matter appeared normal. This partiality of the hemorrhage for the gray matter is very been noticed too often to be considered merely an associated condition, and while in some cases the former may change a predisposition into an active process, in other cases it seems that we must consider it a distinct causal factor. As Schultze has pointed out in a recent article the amount of gliosis in some cases is too limited to permit a supposition that the cavity is due to its disintegration.

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[1] Read at the meeting of the Philadelphia Neurological Society, March 23, 1896.